CURRICULUM VITAE

Name: John Edgar French, Ph.D.

***Position*:** Acting Chief
Host Susceptibility Branch

Division: Division of Intramural Research
National Toxicology Program

Address: National Institute of Environmental Health Sciences
National Institute of Health
P. O. Box 12233
Research Triangle Park, NC 27709-2233
(919) 541-2569; (919) 541-1460 (Fax)

Post-Secondary Education:

1970-1975	Ph.D., Physiology and Cell Biology; North Carolina State University, Raleigh, NC (Mentors: Drs. E. Hodgson and J. Roberts)
1967-1968	Physiology, Colorado State University, Ft. Collins, CO (Mentor: Dr. W. Marquardt)
1965-1967	M.S., Physiology and Immunology; Mississippi State University, State College, MS (Mentors: Drs. B. Glick and R. Sadler)
1961-1965	B.S., Zoology and Chemistry, Mississippi State University, State College, MS

Professional Experience:

2007-Present Acting Chief, Host Susceptibility Branch, National Toxicology Program (John Bucher, Ph.D., Associate Director, NTP), Division of Intramural Research, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina 27709 USA. *Understanding the genetic basis for population-level differences for both toxicants and/or disease susceptibility will lead to a better understanding of how and why substances in our environment are hazardous to some individuals and not to others. Asthma, cardiovascular disease, cancer, diabetes, and obesity are a few examples of diseases associated with multiple interacting genes and their allelic variants that are induced or influenced by environmental exposure to xenobiotic chemicals. The Host Susceptibility Branch) supports the NTP mission by planning, designing, conducting, and analyzing a multi-model assessment of the xenobiotic chemicals that are associated with human diseases or disease processes (e.g., cytotoxicity, tissue regeneration, DNA damage and repair, nuclear receptor-based hormonal signaling, immunosuppression, mitochondrial energetics, xenobiotic metabolism, etc.) resulting from gene-environment interactions.*

2001-2007 Group and NTP Discipline Leader, Transgenic Carcinogenesis, Laboratory of Molecular Toxicology (John Pritchard, Ph.D., Acting Chief), Environmental Toxicology Program, Division of Intramural Research, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina, USA. *Genetically modified rodent models of cancer were used to develop short-term cancer bioassays for the identification of carcinogens and the genes and their allelic variants associated with susceptibility to chemical and physical carcinogens that induce the loss of tumor suppressor genes and tumor progression.*

1996 to 2001 Unit and Discipline Leader, Transgenic Carcinogenesis, Laboratory of Environmental Carcinogenesis and Mutagenesis (Raymond W. Tennant, Ph.D., Chief), Environmental Toxicology Program, Division of Intramural Research, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina, USA. *Genetically modified mouse models of cancer, carcinogen and nutrient gene interactions that modulate the cellular redox state, mitogenesis, and apoptosis were investigated to identify modes of action for human carcinogens.*

1990 to 1996 Research Physiologist, Laboratory of Environmental Carcinogenesis and Mutagenesis (Raymond W. Tennant, Ph.D., Chief), Environmental Carcinogenesis Program, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina USA. *p53 tumor suppressor gene function in chemically induced mutagenesis and carcinogenesis were investigated for the identification of carcinogens.*

1989 to 1990 Senior Scientist, US Public Health Service Foreign Work/Study Fellowship; Combined Nordic Studies, Project on Molecular Genetics, Institute of Occupational Health, Helsinki, Finland. *RAS proto-oncogene mutations and chromosomal abnormalities in asbestos related human lung and pleural membrane tumors were investigated.*

1984 to 1989 Physiologist, Carcinogenesis and Toxicology Evaluation Branch (James K. Selkirk, Ph.D., Chief), Division of Toxicology Research and Testing, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina USA. *Study design, data evaluation and interpretation, and reporting (technical reports/scientific literature) on NTP/NIEHS toxicology and carcinogenesis studies. Management and administration of NTP contracted studies at a major contract laboratory. Technical evaluation and monitoring of study conduct, costs, and laboratory performance by site visit evaluation and reports are essential elements.*

1982 - 1984 Physiologist, Carcinogenesis and Toxicology Evaluation Branch (Bernard Schwetz, DVM, Ph.D., Chief), Division of Toxicology Research and Testing, National Institute of Environmental Health Sciences, NIH, Research Triangle Park, North Carolina USA. *Study director, Carcinogenesis Bioassays; Independent research on chemically induced lesions in the hematopoietic system of rats and mice.*

1978 - 1982	Supervisory Research Physiologist (Blood and Blood Products Branch (Joseph Fratantoni, M.D., Chief), Division of Blood and Blood Products, National Center for Drugs and Biologics, Food and Drug Administration, Building 29, NIH, 8800 Rockville Pike, Bethesda, Maryland USA. *Cell physiology of inflammatory cells and stem cells and the review and evaluation of new drug applications and investigational new drug applications were investigated*.
1976 - 1978	Supervisory Research Physiologist, Department of Experimental Hematology (Sigmund Baum, Ph.D., Director), Radiobiology Research Institute, National Naval Medical Center, Bethesda, Maryland USA. *Research focused on radiation biology and physiology of cell transfusion therapy for immunosuppression, septicemia, and shock.*
1973 - 1975	Post-Doctoral Fellow in Radiation Biology and Toxicology of Mammalian Systems (Sigmund Baum, Ph.D., Mentor), Department of Experimental Pathology, Radiobiology Research Institute, National Naval Medical Center, Bethesda, Maryland USA. *Radiation biology and the dysregulation of cell function in hematopoietic stem cells and inflammatory cells and xenobiotic metabolism.*
1970 - 1973	NIEHS Pre-doctoral Student in Molecular Toxicology and Comparative Biochemistry (Drs. John F. Roberts and E. Hodgson), Inter-Departmental Graduate Program in Toxicology, North Carolina State University, Raleigh, North Carolina USA. *Drug metabolism, reactive intermediates and DNA damage response and replication in prokaryotes and eukaryotes were investigated.*
1968 - 1970	Instructor (Howard Erikson, Ph.D., Chairman), Department of Biological Sciences, Towson State University, Baltimore, Maryland USA.

Honors, Awards and Fellowships:

Visiting Professor, Department of Environmental Sciences & Engineering, School of Public Health, University of North Carolina at Chapel Hill, Chapel Hill, North Carolina, USA, 2004—present.

President-Elect, Genetics and Environmental Mutagenesis Society, 2009

NIH Merit Award, 2008 for development of the NTP Host Susceptibility Initiative

President, Carcinogenesis Specialty Section, Society of Toxicology, 2005—2006.

NIH Merit Award for Genetically Modified Mouse Models for Carcinogenesis Research

Intramural Research Award 1998: Antioxidants and Cancer.

Intramural Research Award 1996: IGF-1 and Urinary Bladder Cancer.

Sustained Performance Award, March 1996; Laboratory of Environmental Carcinogenesis and Mutagenesis, Environmental Carcinogenesis Program (Dr. R. W. Tennant, Chief), NIEHS, Research Triangle Park, North Carolina, USA

US PHS Foreign Work/Study Fellowship, 1989 to 1990, Combined Nordic Ministries Project on Molecular Genetics, Helsinki, Finland (Sponsor: Drs. Richard Griesemer, DTRT, NIEHS and Phil Chen, NIH).

PHS NIH Special Achievement Award, June 20, 1988, for Providing Direction and Guidance in Branch Use of Computers for Meeting Program Goals and Office Automation (Sponsor: Dr. James Selkirk).

Outstanding Employee, Radiobiology Research Institute, National Naval Medical Center, 1977 and 1978 (Sponsor: Dr. S. Baum).

NIEHS Traineeship in Molecular Toxicology and Comparative Biochemistry, 1970-1975, (Mentors: Drs. John Roberts and Ernest Hodgson).

Post-Graduate Training and Instruction in Mammalian Genetics:

Short Course on Medical and Experimental Mammalian Genetics, The Jackson Laboratories, 1988, Bar Harbor, ME

Short Course on Experimental Genetics of The Laboratory Mouse in Cancer Research, The Jackson Laboratory, 1996, High Seas, Bar Harbor, ME

Short Course on Complex Trait Analysis and Statistical Genetics, The Jackson Laboratory, 2007, High Seas, Bar Harbor, ME

Intramural/Extramural Committees/Faculties Service:

NTP Biomolecular Screening Faculty 2009-Present (Kristine Witt, Chair)

NTP/NIEHS Management Committee 2009-Present

NTP Toxicogenomics Faculty 2008-Present (Nigel Walker, Chair)

NIEHS Bioinformatics Search Committee 2008-2009 (Tom Kunkel, Chair)

NIEHS Conferences and Distinguished Lecture's Committee 2009-Present

External Advisory Board for Systems Genetics Program, Oak Ridge National Laboratories, Oak Ridge, TN 2007-Present

External Advisor Board for UNC-CH-MIT Bioengineering Research Partnership in Toxicology, 2007-Present

Planning and Development of the NTP Host Susceptibility Initiative (Lead, October 2006 – October 2007)

Review of the Use of Transgenic Rodent Models for the Environmental Toxicology Program (John Pritchard, Chair)

Transgenic Models Faculty, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

Animal Use and Care (Robert Chapin, Chairman) Division of Intramural Research, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

Career Development and Training (Paul Nettesheim, Chairman) Division of Intramural Research, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

Transgenic Technology and Use Committee (Mitch Eddy, Chairman) Division of Intramural Research, National Institute of Environmental Health Sciences, Research Triangle Park, North Carolina

p53 Model Assay Working Group, *Alternatives to Carcinogenicity Testing Committee*, International Life Sciences Institute, Health and Environmental Sciences, Washington, DC

Special Emphasis Review Panel, CSR, NIGMS, NIH for RO1 Grant Reviews

Society Membership:

American Association for Cancer Research
Complex Traits Consortium
Environmental Mutagen Society
Genetics and Environmental Mutagen Society
Society of Toxicology

Journal Referee

Archives of Toxicology, Cancer Research, Carcinogenesis, Chemico-Biological Interactions, Environmental Health Perspectives, International Journal for Cancer, Nature Genetics, NIEHS Intramural Manuscript Review, Nutrition & Cancer, Oncogene, PLoS Biology, Science, Toxicological Pathology, and Toxicological Sciences

Chair and Organizer, NIH, National, and International Meetings

1. Genetics: The Link Between Exposures, Gene X Environment Interaction, and Toxicity, Society of Toxicology, March 7-11, 2010, Salt Lake City, UT
2. Genetic Susceptibility – The Link between Environmental Exposure and Human Disease, NIH Research Festival, October 14, 2008, Bethesda, MD
3. Host Susceptibility and Chemical Safety Testing: New Approaches to Estimate Risks in The Human Population, Co-Chair with Ivan Rusyn, M.D., Ph.D., 47th Annual Meeting of the Society of Toxicology, March 18, 2008, Seattle, Washington.
4. 5th Complex Traits Consortium, Chapel Hill, North Carolina, Co-Chair with David Threadgill, UNC-CH, Chapel Hill, North Carolina USA, May 6-9, 2006.
5. Alternatives to Carcinogenicity Testing Using Genetically Altered Rodent Models for Carcinogen Identification and Determining Mechanisms of Action, American College of Toxicology, Washington, D.C., November 3, 2003.
6. *p53 and RasH2 Mouse Models*, International Workshop on Genotoxicity, Plymouth, England, June 27-29, 2002.

Invited Symposia/Workshop Presentations: (Reverse Chronology)

1. Genetically Modified Mouse Models for Hazard Identification and Risk Assessment in Toxicology and Carcinogenesis: Strengths and Weaknesses, Society of Toxicologic Pathology, June 23, 2009, Washington, DC

2. Genetic Susceptibility: The Link Between Exposure and Disease, ICCA-LRI Workshop, June 16, 2009, Charleston, SC
3. Genetic susceptibility to DNA strand break damage, loss of heterozygosity (genomic instability), and cancer, NIH Research Festival, October 14, 2008, Bethesda, MD
4. *The radiation induced tumor and DNA strand break repair phenotype in p53 deficient and wildtype mouse hematopoietic stem cells (HSC) in vivo and in vitro is genetic background dependent,* Phenotyping of Model Organisms, CTC/QTRN Satellite Conference, May 31, 2008 Montreal, Quebec, Canada
5. *Host susceptibility and individual susceptibility to environmental agents and diseases,* Host Susceptibility and Chemical Safety Testing: New Approaches to Estimate Risks in The Human Population, Co-Chair, 47th Annual Meeting of the Society of Toxicology, March 18, 2008, Seattle, Washington.
6. *Genetically-Engineered Mouse Models for Hazard Identification and Risk Extrapolation: A Brief Overview on the 20th Anniversary,* CEA-TAC Symposium, 47gh Annual Meeting of the Society of Toxicology, March 17, 2008, Seattle, Washington.
7. *Identification of Presumptive Human Carcinogens and their Mode of Action using Toxicogenetics.* Approaches to determining whether a carcinogen is acting via a genotoxic mode of action, 46th Annual Meeting of the SOT, Charlotte, NC, 25March 2007.
8. *Gene-Environment Interaction in a Mouse Model for Susceptibility to Carcinogen Induced DNA Damage.* Second Annual Conference CRG Unveiling Genome-Wide DNA Variation in 15 Diverse Mouse Strains, September 25, 2006.
9. *Genetic Susceptibility to Ionizing Radiation Induced Loss of Heterozygosity in Mouse and Human Hematopoietic Stem Cells, 5th Annual Complex Traits Consortium Meeting, Chapel Hill, NC, May 7, 2006.*
10. *Signaling pathways for DNA damage and repair, apoptosis, and lymphoid progenitor survival are dysregulated by N-acetyl-L-cysteine*. DNA Evaluation and Repair, Co-Chair and presenter, 44th Annual Meeting of the SOT, New Orleans, LA, March 8, 2005.
11. *Genotoxic stress and genomic instability in the p53 haploinsufficient mouse*. C.L. Davis Symposium, Hoffman-La Roche, Nutley, New Jersey, December 10, 2004.
12. *The role of the p53 haploinsufficient mouse model in toxicology and cancer studies.* American College of Toxicology, Washington, D.C., November 3, 2003.
13. *Alternative carcinogenicity testing with the FVB/N-TgNLed (v-Ha-ras) and the B6;129-Trp53tm1Brd (N5) genetically altered mouse models*. DIA, San Antonio, TX, June 16, 2003.
14. *An overview of selected transgenic models used for carcinogen identification*. NTP Workshop, Washington, DC, February 20, 2003.
15. *p53 and Ras function in cancer*. Japanese Society for Animal Models of Human Disease, Tokyo, Japan, November 6 & 7, 2002.
16. *New Models for Toxicology and Carcinogenesis*. Food Safety Symposium, American Chemical Society, Boston, MA, August 18 & 19, 2002.
17. *Alternative models for identification of potential human carcinogens*. Fourth International Congress for Alternatives, New Orleans, LA, August 14 & 15, 2002.

18. *p53 and RasH2 mouse models for identification of carcinogens*. UK Environmental Mutagen Society, Plymouth, England, July 3, 2002.
19. *The application of mechanistic information from transgenic animal studies for identification and characterization of presumptive human carcinogens.* Workshop on Mechanistic Considerations in the Molecular Epidemiology of Cancer, International Agency for Research on Cancer, Lyon, France, November 14-17, 2001.
20. *The development, characterization, and use of the TSG-p53 haploinsufficient and the Tg.AC (v-Ha-ras) mouse models for rapid identification of carcinogens*. Eighth International Conferences on Environmental Mutagenesis. Shizuoka, Japan, October 24, 2001.
21. *p53 and Hras2 transgenic tumor models working group.* Eighth International Conferences on Environmental Mutagenesis. Shizuoka, Japan, October 22, 2001.
22. *Application of transgenic models in toxicology* (A CE Course Sponsored by the IUTOX). Chinese Toxicology Society, Nanjing, China, October 15 and 16, 2001.
23. *The development, characterization, and use of genetically altered mouse models for identification of carcinogens*. American College of Laboratory Animal Medicine, Point Clear, Alabama, April 3, 2001.
24. *The nature of the p53 haploinsufficient mouse model.* ILSI/HESI Evaluation of Alternative Methods for Carcinogenicity Testing, Leesburg, Virginia, November 1-3, 2000.
25. *p53 Haploinsufficiency, genetic susceptibility, and environmental Carcinogenesis*. Thirty-Eighth Hanford Symposium on Health and the Environment, Richland, Washington, October 19-20, 2000.
26. *Immune suppression and immunomodulation in genetically altered mouse models*. Mechanisms of Immunomodulation, Bethesda, MD September 23, 1999.
27. *Benzene leukemogenesis: an environmental carcinogen induced tissue specific model of neoplasia using genetically altered mouse models*. Benzene State of the Science Workshop, University of Ottawa, Ottawa, Canada, December 15-17, 1998.
28. *Genetically altered mouse models for rapid identification and investigation of carcinogens*. Alternative Toxicological Methods for the 21st Century: Protecting Human Health and Advancing Animal Welfare, Bethesda, MD, December 3, 1998.
29. *Identification and investigation of mutagenic carcinogens using the heterozygous p53 deficient mouse model.* American College of Toxicology, Orlando, FL, November 11, 1998.
30. *Identification and investigation of mutagenic carcinogens using the heterozygous p53 deficient mouse model*. 1st Rodent Models of Modern Risk Assessment Meeting, The Jackson Laboratory, Bar Harbor, Maine, USA, September 8-12, 1998.
31. *Studies in the heterozygous p53 deficient mouse model.* NTP Board of Scientific Counselors, NIEHS, NIH, Research Triangle Park, NC, USA, February 5, 1998.
32. *The use of transgenic animals in cancer testing*. Colloquium on scientific advances and the future in toxicologic risk assessment: 50th Anniversary of the National Research Council's Committee on Toxicology, Washington, DC. USA, December 4-5, 1997.

33. *Genetically altered mouse models for predicting mutagenicity and carcinogenicity.* American College of Toxicology, Washington, DC, USA, November 10, 1997.
34. *The heterozygous p53 deficient mouse model for cancer endpoints in safety assessment.* Drug Information Association Workshop, Noordwijk, The Netherlands, October 26 & 27, 1997.
35. *Transgenic models for mutagenesis and carcinogenesis: testing of xenobiotics*. Eighth Annual North American ISSX Meeting, Hilton Head, SC, USA, October 26-30, 1997.
36. *Applications of transgenic models in toxicology*. Monsanto Annual Toxicology Symposium, Creve Coeur, St. Louis, MO, May 5, 1997.
37. *Evaluation of transgenic mouse models for drug and chemical safety assessment.* Second Annual Conference on Molecular Toxicology, IBC, Rockville, MD, USA, April 28-30, 1997.
38. *Susceptibility of Tg.AC (v-Ha-ras) and p53 deficient transgenic mouse lines to environmental carcinogens identified in long-term bioassays.* US-Japan Workshop: Genetic and Environmental Actions in Cancer, Susceptibility in Animal Models, NIH, Bethesda, MD, USA, March 27-28, 1997.
39. *Update of benzene studies in transgenic mouse models.* The 22nd Winter Toxicology Forum, Washington, DC, USA, February 22-27, 1997.
40. *Rapid identification of mutagenic carcinogens in tumor suppressor gene p53 deficient (+/-) mice.* Barton Creek, Texas, USA, December 4-6, 1996.
41. *Alternative in vitro and in vivo assays using genetically altered mice for predicting carcinogenicity.* American College of Toxicology, Valley Forge, Pennsylvania, USA, November 10, 1996.
42. *Rapid identification of in vivo mutagenic carcinogens using the p53 deficient (+/-) mouse with a lacI neutral reporter gene*. 14th Annual GEMS Fall Meeting, Research Triangle Park, North Carolina, USA, October 11, 1996.
43. *Short-term mutagenesis and carcinogenesis assays in genetically altered mice.* Genetic Toxicology of Pharmaceuticals*,* American Society of Industrial Microbiology, Research Triangle Park, North Carolina, USA, August 6, 1996.
44. *Indirect Mechanisms of Carcinogenesis.* Food and Drug Administration Workshop, Bethesda, Maryland, USA, March 4-5, 1996.
45. *Correlation between mutagenic carcinogens induced mutant frequency and tumorigenesis in* λliz_a*:*$p53^{def}$ *(+/-) F1 transgenic mice*. Transgenic Mice in Mutation Research, Sidney, B.C., Canada, March 20-23, 1996.
46. *Validation of the p53 deficient mouse carcinogenesis model*. 1995 Toxicology Fall Workshop; Pharmaceutical Research and Manufacturing Association, Rockville, Maryland, October 5-6, 1995.
47. *The bi-transgenic lacIq:p53def mouse model for mutagenesis and carcinogenesis studies*. Mini-Symposium on the Use of Transgenic Mice for Short Term Bioassays, National Institute of Public Health and Environmental Protection, Utrecht, The Netherlands, November 10, 1995.
48. *Chemical effects in transgenic mice* In Vivo Transgenic Models, International Congress of Toxicology-VII, Seattle, Washington, July 2-6, 1995.

49. *Short-term chemical carcinogenesis studies in Big Blue ™ TSG-p53 ™ deficient mice.* Midwest Society of Toxicology, Chicago, IL, May 20, 1994.
50. *The p53 deficient mouse model for mutagenesis and carcinogenesis studies. Potential use of transgenic mouse models for in vivo mutagenesis and carcinogenesis studies.* Gene Therapy Symposium, Glass, Research Triangle Park, NC, November 9, 1993.
51. *Chemical effects in transgenic mice*. Environmental Carcinogens and Risk Assessment, Barton Creek, TX, November 30, 1993.
52. *Short-term carcinogenesis studies in mice hemizygous for the wildtype p53 tumor suppressor gene*. Use of Transgenetic Animal Models in Biomedical Research. American College of Toxicology, New Orleans, LA, October 5, 1993.
53. *US NTP summary results on organic solvent toxicology and carcinogenesis studies in rats and mice*. Toxicology of Industrial Chemicals Symposium, 11th Annual Meeting of the Finnish Society of Toxicology, Tampere, Finland, May 25-26, 1990.

Invited Seminars (Selected, Reverse Chronology)

1. Genetic Susceptibility – The Link between Exposure, Toxicity, and Disease, CDER, USFDA, White Oak, MD, October 15, 2008
2. *Genetic Susceptibility to LOH and Cancer.* LPC/LMT Seminars, NIEHS, RTP, NC 27709, October 12, 2006.
3. *Genetically altered mouse models for carcinogens of presumptive risk to humans.* NCEA, USEPA, Washington, DC, January 18, 2005.
4. *Genotoxic stress and genomic instability in the p53 haploinsufficient mouse.* LPC/LMT Seminars, NIEHS, RTP, NC 27709, October 7, 2004.
5. *Genetically altered rodent models and mechanisms of carcinogenesis.* Pfizer, Kalamazoo, Michigan, April 16, 2004.
6. *The role of tumor suppressor gene haploinsufficiency in genomic instability and cancer.* NHERRL, Research Triangle Park, NC, February 25, 2003.
7. *p53 Haploinsufficiency, LOH, and Genomic Instability.* National Cancer Research Center, Tokyo, Japan, November 11, 2002.
8. *p53/Tg.AC/RasH2 transgenic mouse models for carcinogen identification.* IBL, Tokyo, Japan, November 6, 2002.
9. *The application of transgenic mouse models to toxicology and cancer.* University of North Carolina at Chapel Hill, April 24, 2002.
10. *The role of p53 haploinsufficiency in tumorigenesis*. Laboratory of Molecular Carcinogenesis, NIEHS, November 1, 2001.
11. *Use of genetically altered mouse models for pharmaceutical safety assessment*. Glaxo Smith Kline, February 12, 2001.
12. *Potential roles of p53, p16Ink4a, and p19ARF in urinary bladder cancer*. NIEHS, Transgenics Faculty, December 6, 1999.
13. *Use of transgenic mice to identify carcinogens of potential human risk*. Department of Pathobiology, University of Illinois, Champaign-Urbana, Illinois, USA, November, 1999.

14. *Benzene leukemogenesis in transgenic mouse models*. Environmental and Occupational Health Institute, University of Medicine and Dentistry of New Jersey and Rutgers University, New Brunswick, New Jersey, USA, February 19, 1998.
15. *Chemical carcinogenesis in the p53 haploinsufficient mouse*. Chemical Industries Institute for Toxicology, Research Triangle Park, NC, USA, January 5, 1998.
16. *The p53 mouse model*. The National Institute of Public Health and the Environment, Bilthoeven, The Netherlands, October 28, 1997.
17. *The potential for transgenic mouse models in toxicology and carcinogenesis.* NIEHS, Molecular Oncology Faculty, June 9, 1997
18. *The TP53 gene suppresses cancer by regulating critical signaling pathways for DNA damage and repair, apoptosis,* and gene-environment interactions. US EPA, Research Triangle Park, NC, USA, May 20, 1997.
19. *The potential for p53 and Tg.AC mouse models in safety assessment of pharmaceuticals.* Boehringer-Ingelheim Pharmaceuticals Inc., Ridgefield, CT, May 7, 1997.
20. *Carcinogen-induced genomic instability in the p53 deficient mouse*. GD Searle and Monsanto Symposium, St. Louis, MO, USA, May 5, 1997.
21. *p53 Mouse model for cancer*. Triangle Carcinogenesis Club, CIIT, RTP, NC, December 2, 1996.
22. *Conduct of carcinogenesis bioassays using transgenic mouse models*. Integrated Laboratory Systems, RTP, NC, November 22, 1996.
23. *Induction of LOH in the p53 deficient mouse.* Baylor College of Medicine, Houston, Texas, USA, May 10, 1996.
24. *Mutagenesis and carcinogenesis in transgenic mouse models*. Wallenberg Laboratory, Stockholm University, Stockholm, Sweden, May 12, 1994.
25. *Modeling environmental exposures in transgenic mouse models.* Swedish Institute of Occupational Health, Division of Toxicology, February 11, 1994.
26. *Investigation of tumorigenesis mechanisms in transgenic mouse models*. Karolinska Institute, Department of Tumor Biology, Solna, Sweden, December 12, 1993.
27. *Mechanisms of tumorigenesis in the p53 deficient mouse.* Karolinska Institute, Department of Nutrition and Toxicology, Huddinge, Sweden, December 18, 1993.
28. *Mechanisms of mutagenesis and carcinogenesis.* North Carolina State University, Department of Toxicology, Raleigh, NC, September 7, 1993.
29. *Role of folate deficiency in mutation frequency*. University of North Carolina at Chapel Hill, Chapel Hill, NC, September 2, 1993.

Research Fellows

1998–2003 Keith Martin, Ph.D. (IRTA Fellow, Intramural Research Award on Antioxidant/Pro-oxidant Modulation of Apoptosis, Cell Proliferation and Cancer)

1998-2000 Muriel Saulnier, DVM, Ph.D. (Ph.D. student, NCSU-Raleigh)

Student Trainees

2007–Present Rong Jiang, Department of Environmental Sciences & Engineering, Ph.D. student, UNC-CH, Chapel Hill, North Carolina

2006–Present Connie Kang, Department of Environmental Sciences & Engineering, Ph.D. student, UNC-CH, Chapel Hill, North Carolina

2005–2006 Thembekile Dube, Biological Science Laboratory Technician (SIS), Earned BS (Genetics) at NCSU-Raleigh, North Carolina

2003-2005 Matthew Smith, Biological Science Laboratory Technician (SIS), Earned BS (Genetics) at NCSU-Raleigh. Biologist, Cellzdirect, Inc., Pittsboro, NC

1998-2003 Hayden Honeycutt, Matthew Smith, Biological Science Laboratory Technician (SIS), Earned BS (Biochemistry) at NCSU-Raleigh and PharmD. (Nuclear Pharmacy) at UNC-CH. Managing Director, Nuclear Pharmacy, Lubbock, TX.

Internal/External Funding

1-ZO1-ES021207 PI: John E. French Period: 10/01/1995-9/30/2008
Genetic Susceptibility to Loss of Tumor Suppressor Gene Function.
Cancer is a complex disease associated with both inherited and acquired genetic and/or epigenetic changes that modify the outcome from both environmental and endogenous exposures leading to disease. Critical features of cancer include the loss of tumor suppressor gene function and/or activation of proto-oncogenes to oncogenes. These changes are associated with alterations in gene expression that lead to the dysfunction of associated signaling pathways that control the cell cycle, progression, and proliferation of clones of rapidly growing somatic or stem cells that are, most likely, modified by both high and low penetrance genetic variants. Ultimately, this imbalance in gene dosage leads to genomic instability that results in clonal derivation of pre-cancerous cells with uncontrolled programmed cell death and proliferation associated with the development of cancer. These features are common and have been extensively studied in both humans and inbred laboratory rodents as surrogates. The primary aims of this research are to develop improved testing methods for identification of presumed genotoxic human carcinogens and to use mechanistic based studies with inclusion of target-specific, mechanism-based, biological observations to estimate and anchor risk assessment. The general aims of this research are 1) to further the development of genetically engineered mouse models (GEMM) of tumor suppressor gene deficient F1 hybrid mice to identify genotoxic carcinogens of presumptive risk to genetically-susceptible human sub-populations, 2) to identify the genes and their allelic variants that modify the signaling pathways that alter carcinogen potency and tumor incidence in response to environmental exposures, and 3) to use a systems biology and network approach to identify the intersecting nodes of signaling pathways as targets for therapy to mitigate environmental exposure and associated disease.

1-ZO1-ES021134 PI: John E. French Period: 10/01/1994- 9/30/2010
Mechanism(s) of Leukemogenesis in Genetically Altered Mouse Models and Humans.
Public health concern over ionizing radiation and benzene, a radiomimetic, has increased because of their potential to induce hematopoietic cancers, especially in the very young. Early exposure to radiation and benzene may be associated with a depression of blood forming elements leading to anaplastic anemia, followed by lympho- or myelodysplastic phenotypes that ultimately may lead to leukemia or lymphoma. Multiple routes of exposure (Inhalation, oral and/ or dermal) to benzene may results in a slower rate of delivery and a greater internal dose to susceptible target tissues. At equivalent inhalation and/or oral dose (mg/kg/body weight), more benzene may be

expired unmetabolized after administration by the oral route (60%) than by inhalation (14%). Most critical to selection of dose for experimental studies in animal models is the determination of the exposure level that becomes saturating to pathways of detoxification and alters the dose delivered to the hematopoietic stem cell compartment (e.g. bone marrow). For inhalation exposure to mice this is approximately 200 ppm (6 h TWA). Between 5 and 50 ppm benzene (6 h TWA, there is no significant difference between urinary metabolites. Available data also suggests that the ratio of hydroquinone or muconic acid to phenol ratio after inhalation exposure to 50 ppm (6 h TWA) is closer between mice and humans than either rats or Cynomolgus monkeys. This is critical because these may be the most toxic benzene intermediates. Thus, an exposure model benzene induced hematopoietic disease must take into account saturation of benzene metabolism pathways, available data on exposures and high-affinity, low capacity pathways of metabolism that result in the most hematotoxic intermediates. We have been able to show that B6.129-Trp53 haploinsufficient mice exposed to intermittent low levels (100 ppm, 6 h TWA) develop thymic lymphomas rapidly, whereas mice exposed to more frequent and higher levels of benzene (200 ppm, 6 h TWA) develop tumors less rapidly and at a significantly decreased incidence. In these studies, benzene induced lymphomas in the p53 deficient mice were: 1) clonal (T-cell receptor rearrangements were common), 2) showed a pattern of loss or deletions in chromosome 11 carrying the p53 wildtype allele different from sporadic lymphomas, and 3) showed a pattern of dysregulation of critical genes in both the p53 and Rb pathways that affected cell cycle control and population growth and apoptosis. Using hematopoietic stem cell cultures in vitro from B6.129-Trp53 haploinsufficient mice that are homozygous null for the Cyp2E1 gene (critical to activation of benzene to benzene oxide), we have been able to show an absence of toxicity and reduced DNA damage to pluripotential hematopoietic stem cells compared to mice with wildtype Cyp2E1 genes. Using polyclonal rabbit anti-S-phenylcysteine we are developing assays to determine the level of benzene adduction to the bone marrow compartment to determine the relationship between dose and the dose delivered to the target tissue. This will aid in testing the hypothesis that lower doses of benzene metabolized by the stromal cells of the bone marrow and the toxic metabolites produced locally may be directly acting on the hematopoietic stem cell compartment and initiating tumorigenesis.

1-Z01-ES021175 PI: RW Tennant Co-PI JE French 1992-2005

Characterization of epidermal stem cells: biology, carcinogenesis, and function

The skin is a continually renewing tissue consisting of a large population of transit amplifying (TA) cells with a limited proliferative potential, and a smaller population of keratinocyte stem cells (KSCs) that have a high proliferative potential and are clonogenic. KSCs renew the stem cell population and give rise to TA cells, which are displaced to the suprabasal layers and are lost by terminal differentiation. In the skin, the stem cell population resides in the hair follicle bulge, which is located in the permanent portion of the hair follicle and is protected from both physical damage and the changes the hair follicle undergoes as it cycles from resting (telogen) to active growth (anagen). In the well-known two- stage murine epidermal carcinogenesis model, it is a widely held belief that KSCs are the primary carcinogen target cells (i.e., latent neoplastic cells). A key factor supporting this belief is the fact that DMBA-initiated mice will develop skin tumors upon exposure to a tumor promoter such as TPA whether it is applied a week or a year after initiation. Given that the keratinocyte population renews itself every 6 days in the mouse, the fact that initiated cells persist suggests that these must be slowly cycling cells located in a protected microenvironment. Our current focus is on identification and characterization of the cells that give rise to cutaneous neoplasms in the mouse, as well as investigation into signaling pathways that contribute to neoplastic development. As part of this objective, we have investigated the hematopoietic stem and progenitor cell marker, CD34, in the skin and using this marker in combination with alpha-6 integrin and fluorescence activated cell sorting (FACS), have shown

that CD34 specifically marks hair follicle bulge keratinocytes, and facilitates isolation of live follicular bulge keratinocytes that represent a subset of alpha-6 integrin bright cells that are quiescent (i.e., predominantly in G1/G0). This work represents the first use of a bulge-specific cell surface marker allowing for direct positive enrichment of live keratinocyte stem and progenitor cells. Recently we made a comparison was made between CD34+ keratinocytes harvested from either TPA-treated or untreated Tg.AC mice to investigate differential gene expression patterns following tumor promotion. Using nylon cDNA arrays from Clontech probed with PCR-based SMART-amplified cDNA prepared from CD34+ cells isolated from TPA-treated or untreated skin, eleven genes were identified whose expression changed significantly in response to treatment with TPA. Of particular interest was *Deleted* in Split Hand/Split Foot 1 (Dss1), which is associated with a heterogeneous limb developmental disorder. Overexpression of Dss1 and NDPK-B was detected by RT-PCR and Northern analysis in TPA treated skin (non-tumor bearing; compared to low levels in untreated skin), as well as in cutaneous tumors, including papillomas, squamous cell carcinomas, and spindle cell tumors. Functional studies revealed an increase in foci-forming activity and proliferation of preneoplastic epidermal cells constitutively expressing Dss1. Interestingly, Dss1 induced transformation of stably transfected JB6 epidermal cells was abrogated by addition of a protein kinase C (PKC) specific inhibitor, implicating a possible PKC regulatory role in Dss1 expression. Taken, together, these results suggest that Dss1 is a TPA-inducible gene that may play an important role in the early stages of skin carcinogenesis. In addition, recent studies have implicated a potential involvement of NDBK-B in early-stage neoplastic development in chemically induced skin carcinogenesis. In addition, we have furthered these experiments utilizing high density microarray technology, comparing gene expression profiles developed from CD34+ and CD34- cells isolated from TPA treated and acetone control treated Tg.AC and wild type FVB/N mice. From this, comparing back to datasets generated from untreated mice, we have developed a picture of early response genes differentially affected in epidermal stem cells. To understand key signaling pathways in tumor development, we are exploring the relationship between *HRas* and *Cdnk2a* (p19Arf) in benign and malignant tumor development using a bigenic mouse strain made by crossing p19ARF-null and Tg.AC mice. Initial findings in skin tumorigenesis experiments supported a role for p19ARF in tumor development, but in the course of these studies, an unexpected tumor type developed in the bigenic mice, Gastrointestinal Stromal Tumors (GIST), which are highly aggressive tumors in humans, and which developed spontaneously at a high incidence in bigenic mice, making this a potential mouse model for this tumor type. Finally, to assess further the role of the hair follicle epidermal stem cell population in cutaneous tumor development, we have utilized the technique of epidermal abrasion, in which the interfollicular epidermis is physically removed. The resulting epidermal regeneration is derived from keratinocytes migrating out from the underlying hair follicles. Expression profiles using high-density microarray analysis were prepared from samples collected at days 3, 5, 9, 18, and 30 post-abrasion from abraded FVB/N (wild type) and Tg.AC mice (which develop *HRas*-transgene dependent papillomas following a single abrasion). We have developed bioinformatics strategies designed to probe this complex dataset in terms of global analysis, allowing for more informed focus on smaller subsets of genes that may contribute to tumor development and epidermal regeneration.

1-Z01-ES Co-PI J.E. French Period: 3/27/1997-4/1/2000
Carotenoid/Retinoid Modulation of Cellular Redox-Status: The Cancer Cause and Cure Conundrum

The aim of this research is to test a mechanism-based hypothesis that will explain the paradoxical results observed in human intervention trials. It is hypothesized that a cellular environment rich in antioxidants (provided by dietary carotenoids/retinoids) prior to cancer induction will decrease the risk (incidence and severity) for cancer; conversely, the induction of a cellular environment

rich in antioxidants after the appearance of preneoplastic foci will increase the risk and lead to exacerbation of cancer. Studies are proposed: 1) to confirm the ability of select dietary antioxidants (e.g., carotenoids) to modulate the incidence of cancer and associated rates of tumor progression, apoptosis or programmed cell death (PCD), and proliferation in a specific mouse skin tumor model; 2) to evaluate the hypothesis that a dietary increase in antioxidants at two distinct times (prior to or after development of preneoplasia) will have opposite effects on subsequent tumor formation; and 3) to investigate the mode of action of antioxidant associated alteration of tumor progression by determining the putative ability of cellular redox status to influence the homeostatic balance between proliferation and PCD. We anticipate that successful completion of our stated objectives will provide a mechanistic basis to help reconcile the "double-edged sword " phenomenon observed with dietary antioxidants. Recent prevention and intervention trial results imply that consumption of antioxidant-rich foods early in disease states provides a protective advantage, while late intervention with dietary carotenoids/retinoids may exacerbates cancer. We conducted a series of mechanism-based hypothesis driven studies designed to investigate the paradoxical results observed in human intervention trials. We hypothesized that increased antioxidant cellular environment (provided by high levels of dietary supplementation with the chemical antioxidant N-acetyl-L-cysteine or NAC), would alter the cellular redox state and promote cancer in carcinogen-initiated mice. We have observed that high levels of dietary NAC increases survival and reduces multiplicity of skin malignancies but increases the fraction of malignancies induced by topically administered B[a]P. The majority of the malignancies (keratoacanthomas, squamous carcinomas, and spindle cell tumors) induced in FVB/N-p53 deficient Tg.AC mice demonstrated mutant transgene *vHRas* expression and were nucleus positive for p53 (variable intensity from 0 to +4 areas) and treatment independent. Malignancies, both negative and positive for transgene expression were also negative for mutations in codon 12 and codon 61 of endogenous *HRas* protooncogene. NAC alone is mitogenic to splenocytes. In addition, we conducted an in life study (gamma irradiated FVB/N-heterozygous p53+/- mice at 0, 2, 4, or 6 Gy) with and without NAC supplementation in the diet. In this study, we observed a significant decrease in latency for thymic lymphoma and an increased incidence of thymic lymphoma after 24 wks (0/15 vs. 10/15) in the controls versus 4 Gy plus NAC. These results may be explained, at least in part, by in vitro studies that show that lipopolysaccharide (LPS, a B-lymphocyte mitogen) induced splenic cell proliferation (3:1 T- to B-cell ratio) is significantly increased by NAC, but apoptosis was significantly suppressed. Suppression of apoptosis was confirmed by independent assays (TUNEL and Annexin V). Lymphocyte origin specificity was confirmed by flow cytometry using B- and T-lymphocyte specific monoclonal antibodies. We are currently investigating the molecular mechanisms involved in pro-oxidant exacerbation of lymphomagenesis. These studies include changes in gene expression at the mRNA and protein level as well as molecular genetic changes in signaling pathways critical to proliferation and apoptosis. In addition, additional in vitro studies have focused on 1) mechanism of NAC associated suppression of apoptosis using microarray based gene expression, followed by confirmation using protein 2D gel electrophoresis and western analysis (where required for confirming dysregulated pathways).

P42-ES005948 PI: J. Swenberg, Period: 04/01/00 – 3/31/06 NIEHS
Environmental Fate and Effect of Hazardous Chemicals – Project 8 – Quantification and Assessment of Dermal Exposure to Benzene and Naphthalene Using a Noninvasive Sampling of Skin. The purpose of this project is to develop and use a non-invasive tape-stripping technique coupled with analytical chemistry methods to measure dermal exposure to benzene and naphthalene in a selected population of workers. In addition, the potential relationship between dermal exposure and systemic exposure to benzene and naphthalene in these exposed populations will be investigated.

Role: Co-investigator on Project 8

List of Five Significant Publications

- Dunn, SE; Kari, FW; **French, JE;** Leininger, JR; Travlos, G; Wilson, R and Barrett, JC. Modulation of IGF-1 by dietary restriction influences cell death, cell proliferation, and bladder cancer progression in p53 deficient mice. *Cancer Res* 57:4667-4672, 1997. **Role:** Major contributor. Developed the aromatic amine induced bladder cancer model in p53 haploinsufficient mice used and assisted in the study design and analysis of the research. **195 citations (avg. 15/yr)**
- Tennant, RW; **French, JE** and Spalding, JW. Identification of chemical carcinogens and assessing potential risk in short term bioassays using transgenic mouse models. *Environ Health Perspect* 103:942-950, 1995. **Role:** Major contributor. Designed, analyzed, and interpreted the results and wrote the p53 deficient mouse model portion of the studies. **191 citations (avg. 12/yr)**
- Jakubczak, J.L; Merlino, G; **French, J.E.;** Tennant, R; Muller, W; Adhya, S and Garges, S. Analysis of genetic instability during mammary tumor progression using a novel selection-based assay for in vivo mutations in a λ transgene target. *Proc Natl Acad Sci* 93:9073-9078, 1996. **Role:** Significant contributor. Designed and conducted studies for the mutation analysis of the λ transgene and *Trp53* targets in the in wildtype and p53 deficient mouse model and co-authored that section of the manuscript. **131 citations (avg. 10/yr)**
- Dunnick, JK; Herbert, RA; Foley, JF; Seeley, J; Hardisty, JF; Tice, RR; Lacks, G; Furedi-Machacek, EM; Stasiewicz, S and **French, JE.** Phenolphthalein rapidly induces malignant hematopoietic tumors and loss of heterozygosity in the p53 wild type allele in heterozygous p53 deficient (+/-) mice. *Toxicol Pathol* 25(6):533-540 (1997). **Role:** co-author, supervised and participated in the molecular genetic analysis of induced tumors**. (53 citations; avg. 4/yr)**
- **French, JE**; Lacks, G; Trempus, C; Dunnick, J; Mahler, J; Foley, J; Tice, R and Tennant, R. Loss of heterozygosity frequency at the *Trp53* locus in p53 haploinsufficient mice is carcinogen and tissue dependent. *Carcinogenesis* 22:98-106, 2001. **Role:** senior author, designed, and supervised the research. **21 citations (Avg. 2/yr)**

Publications (in preparation and/or review):

- Ke, H; Wei, Q; Parron, V; Waalkes, M and **French, JE.** BCL2 suppresses cell adhesion, apoptosis, and cell proliferation through genetically correlated mechanisms (submitted to Carcinogenesis).
- **French, JE**; Spalding, JW; Hansen, LA; Seeley, J; Trempus, C; Mahler, J; Tice, RR; Furedi-Machacheck, M and Tennant, RW. Benzene induced skin cancer and leukemia in FVB/N-TgN(*v-Has*) transgenic mice (re-submitted to Carcinogenesis 2007).
- Brown, DL; Parron, VI; **French, JE**; Foley, J; Buzzard, J; Kissling, GE; Marion, PL and Cullen, JM. Hepatic and soft tissue neoplasia in HBV transgenic, aflatoxin B1-treated and p53 haploinsufficient mice (accepted Toxicologic Pathology, 2008).
- Ke, H; Parron, VI; Akiyama, SK and **French, JE**. BCL2 functionally interacts with gelsolin to regulate actin polymerization, cell adhesion, spreading, and motility (submitted Oncogene 2008).
- Nylander-French, LA; Kang-Sickle, J-CC; Parron, V and **French, JE**. Dermal exposure

induces keratin adducts in the skin (submitted).

- Kang-Sickle, J-CC; Parron, V; Ball, L; **French, JE** and Nylander-French, LA. PAH induce CYP2E1 and generates napthyl adducts in the viable human epidermis (reconstructed human skin) (submitted 2008).

- Betz, BL; Wei, S-J; Malarkey, DE; Trempus, CS; Humble, MM; **French, JE** and Tennant, RW. Activated H-Ras cooperates with p19ARF deficiency to induce gastrointestinal stromal tumor (GIST) in mice. Gastroenterology (submitted 2008).

Publications (peer reviewed; reverse chronology): (average 56 citations/yr for all publications)

1. Karlson, A; Rasmussen, A; **French, JE** and Söderkvist, P. Notch1 is a frequent mutational target in chemically induced lymphoma in mouse. Intl. J. Cancer, 123(11):2720-4, 2008.

2. Kang-Sickle, J-CC; Fox, DD; Nam, T-G; Jayaraj, K; Ball, LM; **French, JE;** Klapper, DG; Gold, A and Nylander-French, LA. S-Arylcysteine adducts in keratin as biomarkers of dermal exposure to aromatic hydrocarbons. Chem Res Toxicol 21; 852-858, 2008.

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